JACC Vol. 22, No. 4 (Supplement A)
October 1993:20A–1A

Discussion I

BERTRAM PITT, MD, FACC, CHAIRMAN, MARTIAL G. BOURASSA, MD, FACC, WILSON S. COLUCCI, MD, FACC, KALON K.L. HO, MD, MILTON PACKER, MD, FACC, PHILIP A. POOLE-WILSON, MD, FRCP, FESC, THOMAS W. SMITH, MD, FACC, SALIM YUSUF, MBBS, FRCP, DPHIL, FACC

B. Pitt: Dr. Bourassa, there seem to be some differences between the data that Dr. Ho presented on the mortality rate in women as compared with the data in the SOLVD Registry. Because women tend to be older, do the mortality rates decrease if you adjust the SOLVD Registry data for differences in age?

M. Bourassa: In the SOLVD Registry data base, women were at least 5 years older than men, but after adjustment for age, there was still a higher incidence of events in women than in men.

P. Poole-Wilson: How can you explain the very high incidence of hypertension in the Framingham Study? In recent data from Europe the number of cases of heart failure caused by hypertension is much lower than the data from Framingham.

K. Ho: It is difficult to attribute specific causes to cases of heart failure, especially in patients with multiple preexisting medical conditions. In the Framingham Study, the percentage of patients with heart failure who had a history of hypertension is approximately 70%. One of the advantages of the Framingham Study is that we followed up patients for 40 years, and hypertension can be diagnosed at anytime during this 40-year period.

B. Pitt: We were impressed by overwhelming prevalence of ischemic heart disease in the SOLVD Registry data base.

T. Smith: I was intrigued by that same finding. Most of us have taken note of the high incidence of hypertension in the original publication of the Framingham data by McKee et al. (McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham Study. N Engl J Med 1971;285: 1441–6), which differs from the findings in other studies of heart failure. In addition, in the McKee report, there was a 5-year mortality rate of approximately 40% in women and approximately 60% in men. Now you are showing us data that are significantly worse than the rates in the original report. What differences in patient demographics could account for that? Or alternatively, what changes in practice?

K. Ho: The data that I showed you were not age adjusted; they were overall survival rates. In addition, the data that I presented today includes the offspring of the original Framingham cohort, a younger group than the population included in the 1971 report.

T. Smith: That should make things better, shouldn't it? Not worse.

K. Ho: Yes. However, at the same time the original population is growing older and a higher proportion of the original cohort is dying.

S. Yusuf: The data you presented today indicate a prognosis that was about twice as poor as that reported in the earlier report from Framingham. In the SOLVD prevention trial, patients were followed up until they developed heart failure and then were followed up for many years. Even in this group with new onset heart failure, the mortality rate is only approximately 30% at 2 years, a mortality rate that is substantially lower than yours. The only explanation I can think of is that patients older than 80 years were not included in the SOLVD trial.

M. Packer: Some of the differences in mortality among studies of heart failure may be related to differences in how heart failure was defined. The criteria proposed by Dr. Ho are appropriate if one is conducting a large population-based study. In contrast, I believe that the SOLVD Registry primarily focused on hospitalized patients.

Clinicians are heavily influenced by measures of ventricular function. I believe that many physicians ignore the signs and symptoms Dr. Ho has used as criteria for the diagnosis of heart failure when the left ventricular ejection fraction is normal—even though that might not be appropriate. I wonder to what degree the changes you have seen from 1971 to the present are influenced by the increasing use of noninvasive tests. For example, is it possible that many patients given the diagnosis of heart failure in the 1970s had diastolic dysfunction? Such patients might be excluded by physicians participating in epidemiologic surveys carried out in the 1980s and 1990s.

K. Ho: The data that I showed used criteria instituted in the 1940s. We did not use measures of left ventricular systolic function, so the criteria have remained the same for 40 years.

W. Colucci: Do you have measurements of ejection fraction in a subset of patients? Do you have any way of giving us a rough estimate?

K. Ho: There are some direct and indirect data. We have

0735-1097/93/$6.00

performed echocardiograms during the last several examination cycles in the Framingham patients, but we have not measured ejection fraction. Marantz et al. (Marantz PM, Tobin JN, Wassertheil-Smoller S, et al. The relationship between left ventricular systolic function and congestive heart failure diagnosed by clinical criteria. Circulation 1988; 77:607–12) evaluated the ability of clinical criteria to predict ejection fraction in patients with congestive heart failure. On the basis of their population of patients referred for radionuclide ventriculograms, they found that the criteria used in the Framingham Study were 63% sensitive and 63% specific for predicting a left ventricular ejection fraction of ≤40%.